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Do diagnostic segmental nerve root blocks in chronic low back pain patients with radiation to the leg lack distinct sensory effects? A preliminary study

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Background. The present preliminary study documents the effects of a selective nerve root block (SNB) with short or long acting local anaesthetic compared with baseline measurements in patients with chronic low back pain radiating to the leg with maximum pain in one dermatome (L4).

Methods. Ten consecutive patients underwent 20 controlled SNBs at L4 with ropivacaine 0.25% and lidocaine 1% in a prospective, randomized, double blind, crossover fashion. Baseline measurements included sensory function (assessed by pinprick on both unaffected and painful leg) and pain (Verbal Numeric Rating Scale; VNRS, 0–10). A change in size of areas with altered sensory function >10% and a VNRS change of 2 points were considered clinically significant. *P*-values < 0.05 were considered statistically significant.

Results. Asymptomatic hypoaesthesia, variable in extent and non-dermatomal in distribution, was present in seven patients at baseline. It appeared to be more extensive and distal with longer duration of pre-existing pain. SNB produced no consistent changes in extent and distribution of hypoaesthetic areas. Change in VNRS did not correlate with the extent of pre-block or post-block hypoaesthesia. No differences in effects were found between lidocaine and ropivacaine.

Conclusions. Pre-block assessment of sensory function is essential to assess the net effect of SNBs. In this small study group, SNBs failed to demonstrate uniform or distinct effects on sensory function.

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In many patients suffering from chronic low back pain radiating to the leg (CLBP-r) no clear causative process can be identified. Segmental nerve root blocks (SNBs) have been suggested as a means to identify the 'symptomatic' spinal nerve segment and are typically used for diagnosis and prediction of the outcome of surgical or invasive pain treatment.^{1–9} In earlier studies we measured the effects of SNB on sensory¹⁰ and motor function¹¹ and found a large variability in effects. However, insight into the net effect was not obtained as no baseline measurements were made. In view of the altered sensory

processing (neuroplasticity) described in a variety of chronic pain conditions,^{12–15} such alterations may also be expected to be present in CLBP-r patients. Clearly, if sensory function is already altered before SNB, this will influence interpretation of SNB outcomes. To date, however, neither extent nor distribution of pre-existent sensory changes, nor how they are affected by SNB,¹⁶ has been formally studied.

The present preliminary study documents the alterations in sensory function present before the SNB, and the effects of subsequent SNBs on sensory processing.

Patients and methods

Patients were consecutively recruited from referrals to our pain clinic for symptomatic invasive pain treatment. All patients had been examined extensively by experienced neurologists, orthopaedic surgeons or both, including CT, MRI and EMG, and were diagnosed as having chronic low back pain unilaterally radiating beyond the knee. According to our standard hospital protocol such patients are subjected to a series of diagnostic lumbosacral SNBs. Patients with maximum pain in dermatome L4 were included in this prospective, randomized, double blinded, crossover pilot study. Patients were recruited in accordance with the rules of the Declaration of Helsinki. The Hospital Ethics Committee approved the study. Written informed consent was obtained from each patient.

Inclusion criteria were: more than 18 yr of age, pain present for at least 6 months and a verbal numeric rating scale (VNRS; 0 is no pain, 10 is intolerable pain) score of ≥ 5 at the moment of inclusion in the study. Exclusion criteria were: planned surgery, symptomatic neurological deficits, known hypersensitivity to amino-amide-type local anaesthetics or iodide, presence of coagulopathy, or mental disorders. All included patients were scheduled for test blocks with local anaesthetics at spinal level L4. Each patient underwent, on separate occasions, two test blocks with commonly used local anaesthetic agents in random order, one with lidocaine 1% and one with ropivacaine 0.25%, as each other's control to raise the validity of the block response. We assumed the ratio for the relative anaesthetic potencies for lidocaine and ropivacaine to be 1:4.¹⁷ The duration of effect was not a study goal. The hospital pharmacist performed randomization for the first L4 treatment with lidocaine or ropivacaine via sealed numbered envelopes. The second test block at L4 was performed on another day with the other drug.

Three anesthesiologists specialised in invasive pain treatment performed the blocks. A research fellow, unfamiliar with the local anaesthetic agent used, assessed sensory function 30 min before and 30 min after the SNB. The patients' sensation was tested by pinprick (Hypo^R, 825044A, 27G, MPL Technologies Inc., Franklinpark, IL, USA) by 2 cm interval circles from the distal end of the feet up to dermatome T12. The patient was asked to state whether sensation was normal, less or more intense compared with the unaffected leg. Areas with sensory changes, if present, were marked and photographed digitally (Sony MVC-FD7) with a ruler for calibration. Sizes of areas were calculated with a specially developed software program (JvE). A change in area size of $<10\%$ was considered clinically insignificant. Pre-block and post-block VNRS pain scores were also recorded. A decrease in VNRS score ≥ 2 points was considered clinically significant.¹⁸

SNB was performed under fluoroscopic guidance using sensory and motor electro-stimulation with frequencies of 50 and 2 Hz, respectively, for spinal nerve root identification. After visualising the nerve root by using 0.3 ml

contrast dye (Omnipaque[®] 180 mg ml⁻¹; Nycomed Ireland, Ltd, Cork), 0.7 ml of the study solution [lidocaine 1% or ropivacaine 0.25% (Astra Pain Control, AB Södertälje, Sweden) with Omnipaque[®] 15%] was injected. For a more detailed description of the SNB procedure see Wolff and colleagues.¹⁰ All data were initially processed using Microsoft Excel 2000.

Statistical analysis

Changes in size of area with altered sensory and pain VNRS were analysed by Statistica software package (Release 6, Statsoft Inc., Tulsa, OK, USA). Using Mann–Whitney *U*-testing we compared pre-block and post-block areas and pain for the grouping variables lidocaine vs ropivacaine and block order. Comparison of pre-block vs post-block VNRS was performed by Wilcoxon matched pair testing, and pre-block and post-block areas were compared using Friedman's 1-way ANOVA. Relationships between pain and size of areas were tested using Spearman correlation. The relationship between duration of pain and pre-block summed hypoaesthetic areas was assessed by linear regression analysis. *P*-values <0.05 were considered significant.

Results

Ten patients (6 male, 4 female, mean age 47 yr, SD 12, range 25–63) were included to undergo a total of 20 SNBs. Patient characteristics and details of their medical history are presented in Table 1.

Baseline measurements

In seven patients areas with hypoaesthesia for pinprick were found in the affected limb before both blocks, but in three patients no pre-block hypoaesthesia was detected. In none of the patients was hypoaesthesia observed in the unaffected limb. No patients showed hyperaesthesia in the affected or unaffected limb. In all patients hypoaesthetic areas did not correspond to the pain radiation pattern, and showed a non-dermatomal distribution (Fig. 1 and Table 2).

The median pre-block pain VNRS was 5 [interquartile range or IQR 4–7 (range 2–8)]. In three patients, pre-block VNRS scores differed at least two points between first and second session (patients 1, 5 and 9), but for the group as a whole the difference between sessions 1 and 2 was not significant. No major differences were observed in mean sensory or motor electro-stimulation thresholds between the two sessions, but sensory thresholds were significantly lower when the number of painful dermatomes was higher (Spearman $R = -0.56$; $P < 0.05$). There was no relation between the level of electro-stimulation thresholds and pre-block VNRS.

Post-SNB measurements

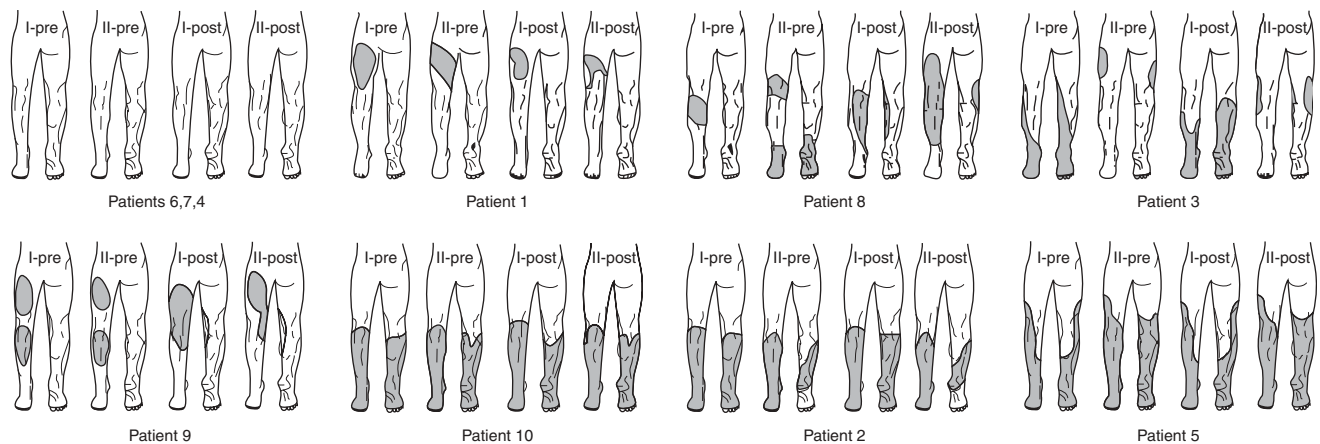
In patients with a baseline hypoaesthesia, SNB produced variable but neither statistically nor clinically significant

Table 1 Patient characteristics and medical history

| Patient | M/F, Age (yr) | Duration complaints (months) | Radiological Diagnosis (MRI, CT, X-ray) | Spinal level | Previous surgery | Medication |
|---------|---------------|------------------------------|---|--------------|--------------------|------------------------|
| 1 | F, 50 | 24 | Facetarthrosis, spondylarthrosis bulging disc, rupture annulus | L5–S1 L4–5 | – | NSAID, codeine |
| 2 | M, 52 | 144 | Facetarthrosis, lateral recess, spinal stenosis epidural fibrosis | L3–4 L2–3 | 2×laminectomy | Acetaminophen |
| 3 | F, 63 | 60 | Lateral facetarthrosis, herniated disc | L4–5 | 4×hernia operation | Acetaminophen |
| 4 | F, 25 | 18 | Bulging disc | L4–5 | – | Acetaminophen |
| 5 | M, 58 | 360 | Lateral recess, spinal stenosis | L4–5 | – | NSAID |
| 6 | M, 53 | 8 | Herniated disc | L3–4 | – | Acetaminophen |
| 7 | M, 40 | 12 | Herniated disc | L5–S1 | – | Acetaminophen |
| 8 | F, 42 | 36 | Bulging disc herniated disc | L4–5 L5–S1 | – | Acetaminophen, codeine |
| 9 | M, 30 | 108 | Herniated disc, discopathy discopathy | L4–5 S1–S2 | Chemonucleolysis | NSAID |
| 10 | M, 55 | 120 | Arthrosis, small foramen, discopathy | L4–5 L5–S1 | – | Acetaminophen |

Table 2 Results

| Patient no. | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|------|-------|-------|------|-------|------|------|-------|------|-------|
| Duration of pain (months) | 24 | 144 | 60 | 18 | 360 | 8 | 12 | 36 | 108 | 120 |
| First SNB | | | | | | | | | | |
| First local anaesthetic | Ropi | Lido | Lido | Ropi | Lido | Ropi | Lido | Ropi | Ropi | Lido |
| Pre-block hypoaesthesia-1 (mm ²) | 253 | 1082 | 495 | 0 | 593 | 0 | 0 | 223 | 1364 | 1271 |
| Post-block hypoaesthesia-1 (mm ²) | 69 | 1085 | 637 | 0 | 591 | 0 | 0 | 394 | 986 | 1489 |
| Δ area (%) | −73 | 0 | +29 | 0 | 0 | 0 | 0 | +77 | −28 | +17 |
| Pre-block hypoaesthesia-1 dermatome | L1–4 | L3–S2 | L3–S2 | 0 | L4–S2 | 0 | 0 | L3–5 | L2–5 | L3–S2 |
| Post-block hypoaesthesia-1 dermatome | L2–4 | L3–S2 | L3–S2 | 0 | L4–S2 | 0 | 0 | L3–S2 | L2–5 | L3–S2 |
| Δ NRS 1 | −3 | 0 | −8 | 0 | −1 | −3 | −4 | 0 | −5 | −1 |
| Second SNB | | | | | | | | | | |
| Second local anaesthetic | Lido | Ropi | Ropi | Lido | Ropi | Lido | Ropi | Lido | Lido | Ropi |
| Pre-block hypoaesthesia-2 (mm ²) | 512 | 653 | 237 | 0 | 1474 | 0 | 0 | 789 | 1014 | 1418 |
| Post-block hypoaesthesia-2 (mm ²) | 210 | 659 | 209 | 0 | 1485 | 0 | 0 | 951 | 1418 | 1420 |
| Δ area (%) | −59 | 0 | −12 | 0 | 0 | 0 | 0 | −59 | 40 | 0 |
| Pre-block hypoaesthesia-2 dermatome | L1–5 | L3–S2 | L3–5 | 0 | L4–S2 | 0 | 0 | L3–S2 | L2–5 | L3–S2 |
| Post-block hypoaesthesia-2 dermatome | L2–4 | L3–S2 | L4–S1 | 0 | L4–S2 | 0 | 0 | L2–S1 | L2–5 | L3–S2 |
| Δ NRS 2 | 0 | 0 | 0 | −2 | −3 | 0 | −3 | 0 | −6 | 0 |
| Summed pre-block areas (mm ²) | 765 | 1735 | 732 | 0 | 2067 | 0 | 0 | 1012 | 2378 | 2689 |
| Summed post-block areas (mm ²) | 279 | 1744 | 846 | 0 | 2076 | 0 | 0 | 1345 | 2404 | 2909 |
| Δ Summed hypoaesthetic areas | −64 | +0.5 | +16 | 0 | +0.4 | 0 | 0 | +33 | +1 | +8 |

**Fig 1** Dermal maps presenting areas with hypoaesthesia for pinprick before and after the two sessions with SNB, grouped per patient. Each group of figures represents, respectively, the measured areas before the first SNB (I-pre), before the second block (II-pre), after the first block (I-post) and after the second block (II-post). Left legs represent the ventral part of the affected leg, right legs the dorsal part.

changes in extent and distribution of hypoaesthetic areas (Fig. 1 and Table 2). There was no group statistical difference in total post-block hypoaesthetic area between session 1 and 2, even when patients without hypoaesthesia were excluded. Hypoaesthesia was absent in the non-affected limb, hyperaesthesia was absent in both legs.

Post-block, median pain VNRS decreased from 5 [IQR 4–7 (2–8)] to 4 [IQR 1.5–5 (0–8)]. Median change in VNRS was –1 [IQR –3 to 0 (–8 to 0)] and was not different between the two sessions. Change in VNRS did not correlate with pre-block pain VNRS or the extent of pre-block and post-block hypoaesthesia. Clinically significant pre-post block decreases in VNRS (≥ 2 points) were found in 8 of the 20 SNB sessions (Table 2).

No differences were found for lidocaine *vs* ropivacaine or for first *vs* second treatment with respect to pre-block and post-block incidence and extent of hypoaesthesia or for changes in pain VNRS.

Discussion

Most patients in this small preliminary study had pre-existing hypoaesthetic areas in the affected limb, not corresponding to pain radiation patterns and non-dermatomal in distribution. This suggests that for correct SNB interpretation, post-block sensory assessment alone is insufficient. The net hypoaesthetic effects of SNBs were neither consistent nor significant, and a clinically significant pain reduction was only found in a minority of blocks.

As far as we know, this is the first time that such effects have been described. Earlier reports^{10 14 15 19} have described pain-induced changes in pain thresholds and motor function in patients with CLBP-r, and in chronic cervico-brachialgia patients.^{20 21} However, these studies provide no information regarding size and variability of hypoaesthetic areas. A clear explanation for the baseline presence of hypoaesthetic areas in CLBP-r patients cannot be given. However, this phenomenon of areas not concordant with known innervation territories of nerve roots is in keeping with reported extraterritorial spread of sensory dysfunction in chronic neuropathic pain patients.²² We have interpreted these areas as non-dermatomal in distribution, although one could also argue that this distribution is perhaps the result of the patients not displaying dermatomes with definite, fixed boundaries. Furthermore, CLBP-r patients should be considered to form a heterogeneous population in which involvement of adjacent spinal levels cannot be excluded. Moreover, pre-block hypoaesthetic areas often differed before blocks in our study, suggesting spontaneous variability in sensory function. Thus, the interpretation of sensory dysfunction and SNB effect on sensory function remains extremely difficult.

Two possible mechanisms may be proposed to explain the presence of these hypoaesthetic areas, namely nerve damage (small fibre neuropathy), inhibitory neuroplasticity or both.

We cannot exclude small fibre neuropathy. The pattern of changes we found is typical for this, with its presentation

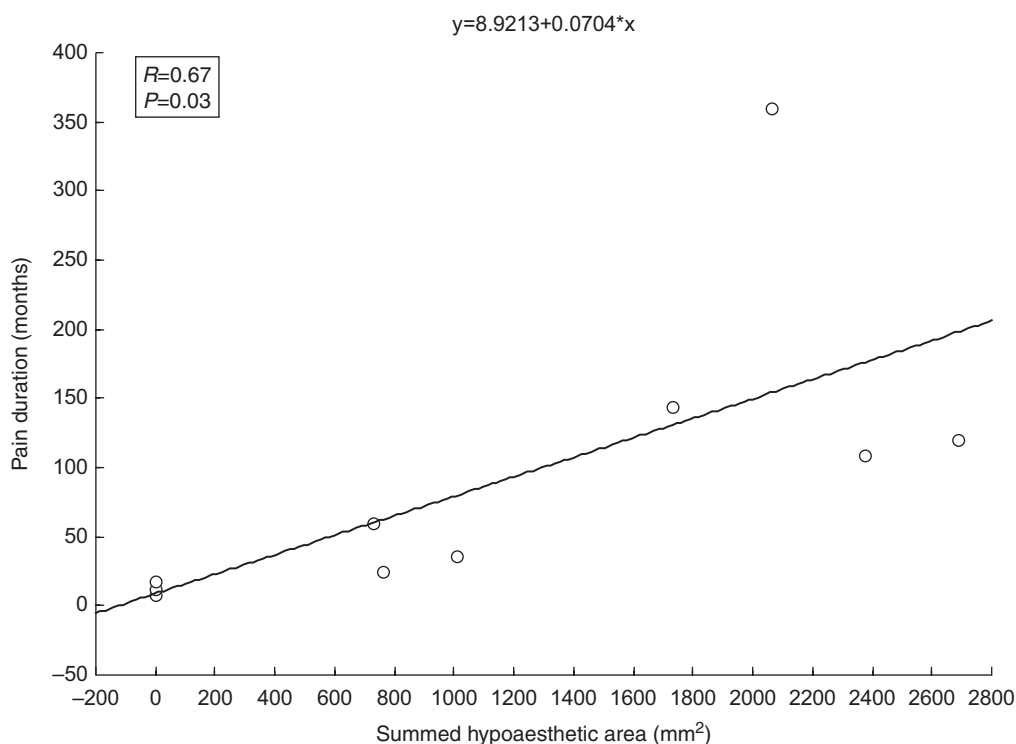


Fig 2 Relationship between duration of complaints and extent of summed pre-SNB hypoaesthetic skin areas ($n=10$).

of pain accompanied by patchy and asymmetrical sensory changes. Pathological processes in the dorsal ganglion, such as demyelination or ion channel re-distribution,¹⁵ are held responsible for this type of small fibre neuropathy,²³ but identification of such processes was not possible in our patients. To formally establish the diagnosis of small fibre neuropathy, more specific complementary diagnostic tests assessing somatic and autonomic fibre system would be necessary.²³

It is well accepted that various forms of neuroplasticity can accompany pain chronification.^{13,24} When we grouped our data according to duration of complaints, the summed pre-block hypoaesthetic areas appeared to be larger in size and more fixed when the duration of pre-existing pain was longer ($R=0.67$; $P=0.03$; Fig. 2). Further support for the involvement of neuroplasticity is found in our observation that electro-stimulation thresholds were significantly lower when the number of painful dermatomes was higher, indicative of pain-induced central sensitisation. Thus it is tempting to postulate that the extent, the variability and the location of the hypoaesthetic areas may be time-related to increasing chronicity of the painful condition. However, considering the small number of patients in the present study one should be cautious. This hypothesis needs to be formally explored further by studying a large population of CLBP-r patients covering the complete spectrum of short to long existing chronic pain. Because pinprick testing alone may miss sensory changes, the use of Quantitative Sensory Testing (QST) in this context offers the possibility of detecting more subtle differences and acquiring more quantified information on the sensory function.

Lack of distinct SNB effect

The lack of a distinct net effect of SNB is surprising in view of the generally assumed axiom that SNB should lead to dermatome related hypoaesthesia and correlated pain reduction. SNB effects may remain unexpressed because of overlap with neighbouring dermatomes.²⁵ Other reasons may include the small number of patients or technical failure, although all blocks were performed under fluoroscopic guidance and were accompanied by clear paraesthesiae and muscle contractions. Furthermore, radiological control demonstrated that in all patients the study drug reached the segmental nerve root L4 without unintended intravascular injection or epidural spread. It is conceivable that a more consistent post-block hypoaesthesia pattern would have been revealed if larger local anaesthetic doses had been used. Complete abolition of intercostal somatic sensory evoked potentials (SSEPs) was reported in thoracic paravertebral blocks using bupivacaine²⁶ in high doses (bupivacaine 0.5% 1.5 mg kg⁻¹). Equivalent inhibition of SSEPs has not been achievable with epidural and spinal anaesthesia.^{27–29} The volume and concentrations of local anaesthetics that we administered were within commonly used equipotent range, although to our knowledge no

controlled dosage-effect studies have been performed up to now in this context.^{9,16} Agents, such as steroids, when added to local anaesthetics in SNBs are also responsible for pain relief³⁰ and can potentiate a local anaesthetic blocking effect.³¹ Our study, however, was aimed at the effects of local anaesthetics only. Differences in pharmacokinetic behaviour between the two study drugs,¹⁶ e.g. differential sensitivity to local anaesthetic agents by different-sized neural fibres, are not addressed by this study but cannot be ruled out. SNB effects might further be attenuated by the inability to block alternative sensory pathways that are part of a multi-segmental neural network.^{32,33} Clearly, our results need confirmation with a larger number of subjects.

Conclusion

In this preliminary study SNBs failed to demonstrate uniform or distinct effects on sensory function. Before the block, asymptomatic hypoaesthetic areas, non-dermatomal in distribution, were observed in many patients. In patients with longer duration of pain, pre-block hypoaesthetic areas tended to be larger. Post-block assessment only must be considered insufficient for SNB assessment, as much of the observed hypoaesthesia was already present before the block. Careful pre-block assessment of sensory function is an essential prerequisite for interpretation of SNB effects.

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